IN THE SPECIFICATION:

Please add the following paragraph at page 13, line 25:

--The mouse cDNA as set forth in SEQ ID NO: 1, encoding mouse SAG (mSAG), was cloned and has been deposited under the terms of the Budapest Treaty at ATCC (American Type Culture Collection, 10801 University Blvd., Manassas, Va. 20110-2209), and bears accession number ATCC 98042. The human cDNA as set forth in SEQ ID NO: 3, encoding human SAG (hSAG), was cloned and has been deposited under the terms of the Budapest Treaty at ATCC (American Type Culture Collection, 10801 University Blvd., Manassas, Va. 20110-2209), and bears accession number ATCC 98045. The human cDNA as set forth in SEQ ID NO: 11, encoding human SAG mutant 1 (hSAG-mutant 1), was cloned and has been deposited under the terms of the Budapest Treaty at ATCC (American Type Culture Collection, 10801 University Blvd., Manassas, Va. 20110-2209), and bears accession number ATCC 98043. The human cDNA as set forth in SEQ ID NO: 12, encoding human SAG mutant 2 (hSAG-mutant 2), was cloned and has been deposited under the terms of the Budapest Treaty at ATCC (American Type Culture Collection, 10801 University Blvd., Manassas, Va. 20110-2209), and bears accession number ATCC 98044.--

Please delete the section "Brief Description of the Drawings" starting at page 4, line 15.

Please replace the paragraph starting at page 21, line 18 with the following amended paragraph:

In order to understand the role of each particular cysteine residues in heme binding and SAG oligomerization, a series of single and double SAG mutants were made in heme binding sites as well as the zinc ring finger motif ~~(see Figure 1B)~~. To generate single point mutations in SAG cDNA, 15 pairs of sense and antisense primers were designed, which are partially

complimentary and contain a desired point mutation. The wildtype SAG cDNA cloned into the pET11a vector at the Nhe I/Bam HI sites was used as the template for PCR amplification. Two separate PCR reactions were conducted using a) primer SAG P.01 (5'-TATGGCTAGC ATGGCCGACGTGGAGG-3) (**SEQ ID 9**) and each of antisense primers and b) each of sense primers and SAG T.02 (**SEQ ID 8**), respectively. The resultant PCR products that overlap with each other and contain a desired point mutation were mixed and served as templates for a third PCR. The primers used were SAG P.01 and SAG T.02, which flank the entire encoding region of SAG cDNA. The PCR was performed as previously described (Sun et al. (1992) BioTechniques 12:639-640). The PCR products were digested with restriction enzymes Nhe I and Bam HI and subcloned into the pET11a vector, which was digested with the same restriction enzymes. To generate SAG double mutants (MM10, MM13, MM14~~, see Figure 1B~~), a QuickChange site-directed mutagenesis kit was purchased from Strategene (La Jolla, CA) and used as instructed. All SAG mutants generated were verified by DNA sequencing (SEQ ID 21, SEQ ID 23, SEQ ID 25, SEQ ID 27, SEQ ID 29, SEQ ID 31, SEQ ID 33, SEQ ID 35, SEQ ID 37, SEQ ID 39, SEQ ID 41, SEQ ID 43, SEQ ID 45, SEQ ID 47 and SEQ ID 49). The predicted mutant SAG proteins encoded by these mutant SAGs are shown in SEQ ID 22, SEQ ID 24, SEQ ID 26, SEQ ID 28, SEQ ID 30, SEQ ID 32, SEQ ID 34, SEQ ID 36, SEQ ID 38, SEQ ID 40, SEQ ID 42, SEQ ID 44, SEQ ID 46, SEQ ID 48, and SEQ ID 50.

Please replace the paragraph starting at page 30, line 30 with the following amended paragraph:

It was next examined whether antisense SAG-transfected cells would exhibit growth inhibition in soft agar. D15-1 cells, along with transfectants expressing wildtype SAG (D12-8), SAG mutant-1 (D3-3), SAG mutant-2 (D4-2), as well as the neo control (D1-3) were grown in

0.25% agar medium for 14 days. Colonies containing greater than 16 cells were counted. Three independent experiments, each run in duplicate, were performed. Shown is the mean +/- standard error of the mean. ~~As shown in Figure 2, down-regulation~~ Down-regulation of SAG in D15-1 cells did cause significant growth inhibition of DLD-1 cells as reflected by 75% reduction of soft agar colony number when compared to the neo control (D1-3), SAG (sense) expressing line, D12-8, and SAG mutants (D3-3, D4-2).

Please replace the paragraph starting at page 31, line 7 with the following amended paragraph:

In a further study, 4 x 10^6 confluent D15-1 cells along with parental DLD-1 cells, the vector control D1-6, and SAG wildtype transfectant D12-1 cells were inoculated subcutaneously into SCID mice (Taconic Farms, Germantown, New York), 10 mice per group. Tumor growth was observed twice a week. The average tumor size/mass for 10 mice was plotted against time post injection up to 24 days. When implanted into SCID mice, antisense expressing line D15-1 failed to form tumors up to 24 days after inoculation, whereas substantial tumor growth was observed in parental DLD-1 cells, the neo control D1-6 cells, and SAG (sense) expressing D12-1 cells ~~(Figure 3)~~. All these experiments demonstrate that downregulation of SAG expression leads to growth inhibition of tumor cells, and further indicates that SAG is a cellular protective molecule.

Please replace the paragraph starting at page 36, line 4 with the following amended paragraph:

To examine whether human SAG can rescue death phenotype of yeast SAG knockout, wildtype human SAG, along with the SAG mutants (MM3, sequence ID 25; MM10, sequence ID 39; and MM14, sequence ID 47, ~~Figure 1A~~) were constructed into a plasmid with Trp selection

marker and transfected into heterozygous yeast strain (y21-SAG/ySAG::Kan) as described above. The clones grown in Trp-minus/G418-plus plates were examined by Western blot analysis for SAG expression. The clones expressing human SAG were sporulated and dissected. In 10 wildtype human SAG clones, 3 or 4 haploids are viable. Some of them contain yeast SAG, whereas the others contain ySAG K/O plus human SAG, indicating human wildtype SAG can complement yeast SAG knockout. All three mutant clones (total of 41 tested) gave rise to 1 or 2 haploids and all survival haploids contains yeast SAG, indicating that human SAG mutants cannot complement yeast SAG knockout.